Module III: Toxicology: Major Substances Affecting the Delta

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Competency Statement

This unit provides an overview of the major toxic substances found in the Mississippi Delta Region (MDR). Descriptions of toxic substances, their properties, processes of emission, and relationships to major health problems are included. The learner will be provided with an abbreviated scientific basis for recognizing the need for nursing intervention. The student will learn how to recognize those agents which have the potential for causing alterations in the health of residents in the MDR. Some agents have insidious (slowly developing), cumulative adverse effects that increase morbidity, while other agents cause rapid death due to the nature of the agent or through synergism with other chemicals in the environment.

Rationale

Health professionals, especially nurses, are witnessing the increasing relevance of toxicology to professional practice. Nurses must become aware of concepts of environmental exposure and consider the possibility of exposure when clients present with signs and symptoms or when events or morbidity or mortality facts and statistics suggest the presence of toxic materials and environmental hazards. Environmental health competencies for nurses include knowledge of the relationship between individuals or populations and environmental hazards as well as responses to toxic exposures (Pope et al., 1995). Many of the basic concepts of toxicology, such as action, absorption, metabolism, distribution, and elimination are familiar to nurses from the study of pharmacology. An understanding of the basic concepts of toxicology is essential in the assessment of risk associated with exposure to hazardous substances at work and in the community.

There are about 200 chemicals that have been identified in the MDR (Nathan et al., 1997) that have an impact upon the life of the residents. Many of the chemicals cause physical and psychological conditions that could be prevented or ameliorated if the properties of these chemicals were better understood by nurses. Nurses are in a position to foster preventive health care practices because of their relative accessibility to residents in communities. Nurses need to be aware of the substances that pollute the external environment of air, soil, and water as well as the home environment. Major chemicals and toxic substances found in the MDR will be examined in this module so that nurses in the MDR and elsewhere will improve their understanding of those substances that have an adverse health effect on community residents.

Objectives

Upon completion of this module, the learner will be able to:

1. Discuss toxicology and its scope of practice in environmental health.

2. Describe the processes toxicants undergo within the body.

3. Describe six hazardous substances that have the potential for causing physical

and psychological illness.

4. Describe the emergency treatment of individuals exposed to various toxic substances.

Vocabulary

Absorption
Antidote
Biodegradability
Biotransformation
Carcinogen
Chelation
Congener
Corrosive
Excretion
HAZMAT
Herbicide
Hydrocarbon
Metabolism
Pesticide
Solvent
Termiticide
Toxic effect
Toxicology
Toxicology, analytical
Toxicology, clinical
Toxicology, environmental
Toxicology, forensic
Toxicology, mechanistic
Toxicology, occupational
Toxicology, regulatory

Course Content

1.1 Define toxicology

Toxicology is the study of the nature and mechanism of toxic effects of substances on living organisms and other biological systems. In addition, toxicology deals with quantitative assessment of the severity and frequency of these effects in relation to the length of exposure of the organism to the toxin (Lu, 1991). Toxicology helps health care professionals to recognize the manner by which chemicals and waste products affect the health of individuals by causing subacute and acute illnesses.

1.2 Discuss toxicology and its scope of practice

Toxicology is a discipline which is used in medicine for diagnosis, prevention, and treatment. In agriculture, toxicology is used to study pesticides, growth regulators, artificial pollinators, and animal feed additives. In the food industry, toxicology is used to study the effect of direct and indirect additives. In the chemical industry, toxicology deals with solvents, components, and intermediates of plastics as well as the effects of metals, petroleum products, pulps and paper. Toxicology is also used to study plant and animal toxins.

1.3 Differentiate the subdisciplines of toxicology

Toxicology involves many subdisciplines. Which subdiscipline is used depends on the living organism involved, the effect of the chemical upon the body, and the form and length of exposure. The subdisciplines are as follows:

Analytical toxicology

Identification of the toxicant through analysis of body fluids, stomach contents, excrement, skin, or suspected containers. This type of toxicology is often used following epidemics or

serious outbreaks of illness.

Clinical toxicology

Administration of antidotes to counter a specific toxicity, lessen or improve signs and symptoms, and promote or quicken excretion of the toxicant from the body.

Forensic toxicology

Study of the legal implications related to the exposure to toxicants and their detection.

Occupational toxicology

Study of intoxication resulting from exposure to toxicants in the work setting.

Environmental toxicology

Study of sources, transport, degradation, and bioconcentration of toxins in the environment and their effect on humans.

Regulatory toxicology

Establishment of standards, regulations, ordinances, and laws to limit or suspend use of toxic chemicals as well as define the conditions of use of various chemicals.

Mechanistic toxicology

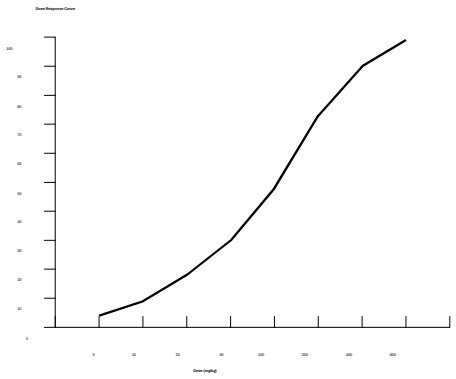
Study and knowledge of the mechanism of action of chemicals.

1.4 Recognize the relationship among factors affecting/causing toxicity of substances

The toxicity of a substance depends on a variety of factors: dose, duration and route of exposure, shape and structure of the chemical itself, and individual human factors.

Dose

A dose is the amount of a contaminant to which



a person is exposed over a period of time. (ATSDR, 1993a). The amount of exposure and the type of toxin to which one is exposed will determine the toxic effect. One of the foundational concepts of toxicology is the dose-response relationship. A dose-response relationship exists when an increase in dose is followed by an increase in response. In other words, the larger the dose, the greater the percent of the exposed population affected. The amount of the dose

(Adapted from: Klaassen et al. [1996]. Casarett and Doull's Toxicology. The basic science of poisons. 5th ed.)

administered per unit of body weight will determine the extent of toxicity. Dose-response curves are indicated by plotting data showing the increasing doses on the x axis and percent response (e.g. increasing mortality) rates on the y axis. (See figure above.)

The slope of the curve provides an indication of the margin of safety of a substance (e.g. the

steeper the slope of the curve, the lower the margin of safety). A steep slope indicates that a large proportion of the population will be at risk from a small range of doses (Ballentyne, 1992). While making a determination of a toxic dose, toxicologists use a measurement of individual body surface area to adjust for variations in leanness and body fat as well as weight of the individual.

Toxicology uses several terms to describe the extent of non-carcinogenic toxicity. The LD_{50} (Lethal Dose $_{50}$) is the dose of a substance that kills 50% of the animals tested over a given period of time. This dose is a frequently used reference point for consideration of lethal toxicity. The lower the LD_{50}, the greater the toxicity of a substance. The NOEL value (No Observed Effect Level) is the highest dose of a substance at which no observable effect is seen in a species. In estimating the NOEL value, it is often assumed (Klaassen et al., 1996) that humans are more sensitive to substances than animals and an adjustment is made in the calculation of levels for humans. The NOEL value can be used as a basis for assigning safe levels for exposure. The LOEL value (Lowest Observed Effect Level) is the smallest dose of a substance that causes any detectable effect.

A toxic dose for a child is less than one for an adult. The threshold value is the dose below which a safe level exists and above which effects are seen (Winder et al., 1997). It is assumed that there are no threshold values for carcinogens that cause mutations (Klaassen et al., 1996). Caution must be exercised in interpreting the above values. All of the values should be interpreted in terms of the conditions of the testing. Toxicologists use multiple sources of data, rather than a single source, to make a determination of toxicity.

Duration of Exposure

Exposure occurs when there is contact between a human being and an environmental contaminant of a specific concentration for an interval of time (ATSDR, 1993a). The duration of the exposure to the toxic substance greatly influences the toxic effect. Repeated daily exposure can cause varying degrees of effect. For example, exposure to a minute or low dose of a toxin over a short period of time may have no effect or little effect; however, daily exposure to the same toxin for a long duration may produce a toxic effect resulting in severe illness or even death .

Route of exposure

The route of exposure influences the toxic effect because the route is an important determinant of the dose amount that enters the body (i.e., some substances are well absorbed by one route but not by another). The three main routes of exposure to environmental toxins are inhalation, ingestion and dermal contact. The respiratory tree consists of the upper airway (nose and pharynx) and the lower airway (trachea, bronchi and alveoli). The upper airway admits air and prepares it for the lower airway by warming it and removing some particulate matter from inspired air. The trachea permits passage of air and continues to remove particulate matter from entering the small airways where gas exchange occurs across the pulmonary capillary membrane.

Inhalation of toxic substances may cause systemic or local effects. Systemic effects following inhalation are caused by the toxicant being absorbed rapidly into the circulating blood and then transported to all parts of the body. Local effects following inhalation may cause damage to the respiratory tree and lungs.

Damaging or harmful effects on the lung parenchyma may be local irritation, edema, allergic response, fibrosis, cellular damage, emphysema and carcinoma. Effects on the respiratory tree may include hyperplasia, ulceration and carcinoma.

The gastrointestinal tract permits the entry of toxic substances when such substances are ingested or when secondary ingestion occurs through hand-to-mouth contact or through swallowing mucociliary sputum that was contaminated in the upper airway. The upper gastrointestinal tract (mouth, pharynx and esophagus) is composed of mucous membrane, a submucosa, a muscular layer and a serous layer. The functions of the mucous membrane are protection, secretion and absorption. The pharynx comprises skeletal muscle with mucous membrane lining. The mucosa of the pharynx contains mucous-producing cells that provide fluid to facilitate the passage of the material swallowed. The esophagus contains muscular tissue that helps propel ingested substances forward by means of peristaltic waves. The stomach begins the process of digestion and prepares substances for absorption in the small intestine. In the stomach small quantities of water, weak acids, glucose, some salts, alcohol and various lipid soluble substances may be absorbed. Most of the absorption of substances in the gastrointestinal tract occurs in the small intestine due to its large surface area, thin diffusion surface and high blood flow (Eaton & Robertson, 1994).

Ingestion of corrosive toxic substances such as strong acids usually cause immediate injury in the form of stomatitis in the upper gastrointestinal tract (i.e., mouth, pharynx and esophagus). Though a low concentration of a toxic material would rarely cause injury to the squamous epithelium of the esophagus, ingestion of higher concentrations can cause injury to the squamous epithelium of the esophagus through the destruction of the epithelial cells. This injury can result in scar formation in the esophagus.

The skin provides an external protective covering for the body and retards the loss of water from the deeper tissues. The skin consists of two layers of tissue, the epidermis and the dermis. Toxic materials absorbed through the skin may cause irritation, chemical burns, and pruritus when they come into contact with the skin. Dermal exposure is one of the routes for systemic exposure.

Shape and Structure of Chemicals

The shape of a molecule enables the chemical to attach itself to other molecules. Subtle differences in a chemical's structure may determine whether a compound is of low or high toxicity. The type, number and placement of groups (e.g. methyl, hydroxyl etc.) on the benzene ring determines the nature and property of the chemical.

The degree of polarity of a chemical often determines the degree of toxicity. Since the human body can efficiently excrete polar substances, the extent of toxicity is decreased when exposure to polarized chemical substances occurs. However, there are exceptions such as heavy metals which are both polar substances and toxic.

Individual Human Factors

Individual human factors play an important role in determining toxicity following exposure to a toxin. One needs to be mindful that very young individuals and the elderly may experience a significant toxic effect from a dose that, in the

average adult, would be a low dose. Immature liver function in children causes impaired metabolism of toxic substances while decreased kidney function in the elderly causes reduced elimination and subsequent accumulation of toxic substances in the system. The size of the individual affects the intensity of the response following exposure to a toxin. Body fat affects the distribution and concentration of a substance at the site of action. Individual variation in specific enzyme activity influences drug metabolism and, therefore, the susceptibility of the individual to toxicity (Blain, 1994).

Toxicological Interactions

Just as with medications, exposure to multiple chemicals can result in biological interactions that may be additive, synergistic, antagonistic or potentiating. Because communities are usually exposed to mixtures of environmental pollutants, nurses should be aware that interactions are possible.

2.1 Describe the major chemical processes that produce reactions within the human body

The kinetics or movement of toxic substances in the body involves:
1) absorption (process of substances entering the body as a result of passive diffusion, facilitated diffusion, active transport or the formation of transport vesicles [pinocytosis and phagocytosis]); 2) distribution (process of the transportation and circulation of absorbed materials in the free state or bound to proteins or blood cells for storage in adipose tissue and bone); 3) metabolism (process of conversion or biotransformation of substances in the liver by means of hydrolysis, oxidation, reduction, and conjugation to produce more water-soluble [polar] products that are capable of removal by urinary excretion); and 4) excretion (process of elimination of a substance from the body as a parent compound, metabolite, or conjugate in the urine, bile, feces, expired air, sweat, hair, nails, saliva, and breast milk).

The action or dynamics of toxic substances in the body depends on the concentration or amount of a substance at the site for action. A substance is available for action if it reaches its site of action in the proper form at the cell membrane. At the cell membrane, permeability of the membrane depends on the molecular size and shape, solubility, degree of ionization, and lipid solubility of the substance.

The rate of absorption of a toxic substance depends on its concentration and solubility. Small water soluble molecules (such as ethanol) are absorbed readily through the aqueous pores of membranes. It is important to remember that most fat-soluble toxic chemicals can readily pass through the lipid part of the cell membrane and be absorbed systemically. Lipid soluble substances are readily stored in fat. Body fat acts as a reservoir with the capability of slowly releasing toxic substances over time (Becker & Rosenberg, 1990, p. 132).

Biotransformation of ingested substances takes place chiefly in the liver. Hepatic microsomal enzymes, as well as extrahepatic enzymes in the skin, lungs, kidneys, and skeletal muscle, play a role in the formation of metabolites of toxic substances. The microsomal enzyme system of the liver breaks down substances to a form whereby they may be absorbed in the small intestine and eliminated from the body through the bile and feces. The most important enzyme in this system is cytochrome P450.

Biotransformation or metabolism by tissue

enzymes causes the breakdown of some substances into less toxic metabolites. On the other hand, it causes a transformation of other substances into more toxic metabolites. Individual differences among humans in the level of activity of the microsomal system account for variation in the response to toxic substances. Genetics, sex hormones, disease state, and developmental level are influencing factors. Immature liver and kidney function in neonates cause a low level of activation of the hepatic enzyme system and renal excretion, which increases the possibility of toxicity.

Biotransformation is a process that often facilitates the elimination of an agent by making it a more polar (water soluble) metabolite. Phase 1 reactions include oxidation, reduction, and hydrolysis. In phase 1 reactions, a polar reactive group is introduced into the molecule to make it more water soluble and suitable for phase 2 reactions. Phase 2 reactions produce compounds (conjugates) that have been synthesized from the toxicant or its metabolite and an endogenous metabolite. Phase 2 reactions add polar biomolecules to a substrate to produce polar metabolites that are easily excreted in the urine. Together phase 1 and phase 2 metabolism converts lipophilic (fat soluble) substances to more hydrophilic (water soluble) products and encourages excretion by certain pathways but primarily through urinary elimination (Liebler & Sipes, 1992, p. 32; Blain, 1994, p. 68).

3.1. From the list of chemicals found in the Mississippi Delta Region, describe commonly found chemicals in the various states, their use, signs, and symptoms of toxicity and emergency treatment

As discussed above, health risk depends on the intrinsic toxicity of the chemical, its concentration, the duration of exposure, and the health status of the person exposed. Diagnosis and treatment information is

known for more acute than chronic exposures.

Chronic exposure, which refers to repeated exposures over a period of time, often produces health effects that differ in type or degree from effects of acute, short-term exposure. Environmental exposures are often long term and low level with a prolonged latency period before symptoms appear.

Generally, acute toxicity scenarios due to high dose exposures will tend to present with more specific symptoms that are clues to the origin of the symptoms and facilitate diagnosis whereas those with chronic, low dose exposures will likely present with more nonspecific symptoms and make diagnosis more challenging.

Example:
Symptoms of Organophosphate poisoning

- The Acronym MUDDLES (Miosis, Urination, Diarrhea, Diaphoresis, Lacrimation, Excitation, Salivation) is used to describe the cluster of symptoms from the autonomic nervous system (AN) and CNS that can occur with acute exposure. **Miosis** (small pupils) in combination with nausea, confusion and fasciculations alert the physician to the possibility of acute organophosphate poisoning.

- Chronic low dose exposure may have symptoms that are flu-like in nature: headache, nausea, vomiting and fatigue.

Acetone (Dimethyl Ketone)

a. Description

Acetone is a colorless liquid with a distinct smell and taste. It evaporates easily, dissolves in water, and is highly flammable and a fire hazard. A fire caused by an acetone spill will produce poisonous gases. It occurs naturally in plants, trees, volcanic gases, and forest fires and is a product of the breakdown of body fat. It is also released during its manufacture and use, in exhaust from vehicles, tobacco smoke, and landfill sites and certain kinds of burning of waste materials. Industrial processes contribute the majority of acetone to the environment. During its manufacture, acetone is released into the air with approximately one-half of the chemical broken down. Rain and snow move acetone to soil and water. Although it does not bind to soil, acetone can move into ground water from spills or landfills.

The major source of exposure to acetone occurs in workplaces where workers who deal with paints, plastics, artificial fibers and shoe manufacturing are exposed to higher levels of acetone. Smoking or breathing secondhand smoke also exposes individuals to acetone. Exposure may also occur by direct skin contact, and by ingesting water or food that contains acetone. Children who eat soil that is contaminated by a landfill or hazardous waste site that contains acetone may also be exposed.

b. Uses

Acetone is used as a volatile solvent (in fingernail polish remover, glues, and rubber cement) and as a defatting agent in the semiconductor industry.

c. Signs and Symptoms

Acute acetone toxicity causes the release of norepinephrine in the heart and lungs causing respiratory and central nervous system (CNS) depression. Symptoms of exposure to moderate or high levels of acetone can cause nose, throat, lung, and eye irritation; shortening of the menstrual cycle; headaches; light head; ataxia; hypoglycemia; hyperglycemia, acetonuria; confusion; increased pulse rate; blood clotting effects; vomiting; seizures (children); hypotension, lethargy; unconsciousness; and coma. Swallowing very high levels of acetone can cause unconsciousness and damage to oral mucosa.

Chronic dermal contact can cause skin irritation (dermatitis) and dryness.

d. Emergency Treatment

Emergency treatment for inhalation injuries from acetone includes removing the person from exposure and assessing the need for rescue breathing or cardiopulmonary resuscitation (CPR) while awaiting transfer of the victim to an emergency room. If exposure has occurred through spillage on the skin, remove any contaminated clothing and wash contaminated skin with large amounts of soap and water. For eye contact immediately flush the eye with large amounts of water for at least 15 minutes. Do not induce vomiting in the case of ingestion of acetone. Activated charcoal may be given to absorb some of the acetone from the gastrointestinal tract. Biomarkers of recent exposure are expired air, urine and blood (ATSDR, 1994d; Leikin & Paloucek, 1998; New Jersey Department of Health, 1992).

Ammonia

a. Description

Ammonia is a colorless, highly irritating gas with a pungent, suffocating odor. This volatile alkaline gas is formed by the breakdown of nitrogenous substances. It dissolves readily in water to form ammonium hydroxide, a corrosive, alkaline solution. Routes of exposure to ammonia include inhalation, skin/eye contact, and ingestion.

b. Uses

Ammonia is used in commercial fertilizer, industrial refrigerant, explosives, petroleum refining, and in other chemicals. Ammonia and hypochlorite form chloramine, which is sometimes used as a disinfectant in water purification. Ammonia is also found in many household and industrial cleaning products.

c. Signs and Symptoms

Ammonia fumes may cause headache, lacrimation, blurred vision and severe eye pain. Exposure to ammonia gas or ammonium hydroxide can result in corrosive injury to the skin and the mucous membranes of the eyes, lungs, and gastrointestinal tract. Exposure to concentrated ammonia vapors cause mucosal burns to the eyes, nose, pharynx, and larynx. Eye exposure may result in conjunctivitis, lacrimation, corneal irritation and temporary or permanent blindness. Respiratory symptoms of upper airway irritation, dyspnea, chest pain, bronchitis, pulmonary edema nausea and vomiting.

Chronic exposure to ammonia may cause bronchiolitis, pulmonary fibrosis, chronic obstructive pulmonary disease and asthma. Airway hyperactivity has been noted in several case reports. Chronic irritation of the conjunctiva also has been reported.

Ammonia is not considered to be carcinogenic. At doses that do not cause maternal toxicity, ammonia is not likely to have adverse reproductive and developmental effects. If maternal pulmonary function becomes severely compromised because of irritation or corrosion, there is a possibility of nonspecific effects on the unborn.

d. Emergency Treatment

Remove the victim from the source of exposure while awaiting transport to the emergency room. Exposed eyes may be irrigated with copious amounts of water. For dermal exposures, wash the exposed area throughly with soap and water. In case of ingestion, do not induce vomiting or perform gastric lavage. Stomach contents may be diluted with water or milk. Steroids may be administered for esophageal burns. Observe for pulmonary edema (Leikin & Paloucek, 1998; ATSDR, 1994a; Haddad et al., 1998).

Arsenic

a. Description

Arsenic is a grayish white solid with a metallic taste and the odor of garlic. It is commonly found as arsenic trioxide, arsenic trichloride, sodium arsenate, arsenic pentoxide, arsenic acid, and other arsenates. Minute harmless traces are found in vegetables, eggs, animal meat, and some seafood. Arsenic is mainly transported from industrial contamination by air and by water. Environmental sources of arsenic exposure are contaminated food, water, soil and air. Ingestion and inhalation are the

primary routes of entry.

b. Uses

Arsenic compounds are used as pigments and refining agents in glass, tanning and taxidermy. Commercially, arsenic is used in the manufacture of household and garden rodenticides, pesticides, and herbicides. Arsenic is used for purifying industrial gases in the smelting industry, in the manufacture of electronics , and lasers. Certain types of arsenic are used in the manufacture of calcium, copper and pesticides. It is also used in homeopathic remedies, especially in Asian cultures.

c. Signs and Symptoms

Arsenic poisoning causes multisystem injury. By inhibiting oxidative phosphorylation enzymes, arsenic toxicity may lead to damage of the liver and kidney, and to the gastrointestinal, cardiovascular, neurological, respiratory, and hematopoietic systems.

Symptoms of acute arsenic poisoning commonly appear within one hour but may be delayed for several hours. Ingestion causes garlic odor of breath and feces; damage to the mucous membranes of the eyes and nose producing irritation, vesicle formation and tissue sloughing, copious blood tinged diarrhea, nausea, vomiting, abdominal pain, cold, clammy skin, muscle cramps, facial edema, proteinuria, hematuria, glycosuria, oliguria and acute tubular necrosis. Shock, cyanosis, and cardiac dysrhythmia (torsades de pointes), jaundice, anemia, leukopenia, and thrombocytopenia may be apparent. Seizures, coma and circulatory failure may result in death.

Chronic occupational exposure to arsenic is rare. Deliberate long-term poisoning has been reported. Chronic arsenic poisoning from repeated absorption of arsenic produces a slow onset of manifestations of dermatitis; hyperpigmentation; symmetric hyperkeratosis of the palms of the hands and soles of the feet; and edema of the face, eyelids, and ankles. Arsenic concentrates in hair and nails are evidenced by Mee's lines (transverse white lines appearing above the lunula of the fingernails about five weeks after exposure to arsenic). Neurologic effects include peripheral neuropathy (paresthesia, pain, anesthesia, ataxia) and hearing and sensorimotor weakness or loss. Other symptoms include vomiting, abdominal pain, thirst, bloody diarrhea, dehydration, fever, anorexia, hepatomegaly, anemia, and electrocardiogram abnormalities.

d. Emergency Treatment

Emergency treatment of acute exposure to arsenic involves rapid replacement of fluids and electrolytes, instituting gastric lavage, inducing emesis with activated charcoal and sorbitol, hemodialysis, and chelation therapy. The antidote for arsenic poisoning is British Anti-Lewisite (BAL, dimercaprol), a chelating agent that acts to bind and remove arsenic from the body. DMPS (2,3-Dimercaptopropane - sulphonate) has been shown to prevent polyneuropathy if given within 48 hours of exposure.

Treatment of chronic exposure focuses on identification of the toxic source and supportive measures to minimize discomfort and manage symptoms (Leikin & Paloucek, 1998; ATSDR, 1990a; U.S. EPA, 1989). Exposed individuals require follow-up to monitor liver, renal and blood studies (Haddad et al., 1998).

Asbestos

a. Description

Asbestos is a family of silicate minerals that occur naturally in some soil and rocks. The fibrils of individual tubes of single crystals are bound together to produce fibers of high tensile strength, flexibility, and durability. Asbestos is non-biodegradable and environmentally cumulative. It is soluble to varying degrees in acid solutions.

Asbestos is dispersed into the air and soil from the weathering of natural deposits but, primarily, from the demolition, renovation, repair, and maintenance of commercial or public buildings . Exposure occurs by breathing contaminated air in workplaces that make or use asbestos or in homes where asbestos was used for insulation around pipes or in ceiling tiles. Asbestos exposure can also occur from breathing contaminated air near asbestos waste or ingesting contaminated drinking water. Asbestos is not biotransformed and remains permanently in the body. Because asbestos is a human carcinogen, it is regulated by EPA and OSHA. OSHA has set a limit of 0.2 fibers/ml over an 8 hour workday and a 30 minute maximum of 1 fiber/ml.

b. Uses

Asbestos is used primarily in insulation of buildings, pipe insulation, boiler coverings, floor and ceiling tiles, fire proof clothing, break linings, transmissions, friction products, and in locomotive repair and power plants.

c. Signs and Symptoms

Asbestos is a respiratory irritant causing a condition called asbestosis. Signs and symptoms of chronic asbestos exposure include shortness of breath and cough, rales, skin warts, renal failure, and leukemia. There is a long (many years) latent period between time of exposure and illness. Inhalation of very high levels of asbestos fibers can lead to asbestosis with interstitial fibrosis of the lung parenchyma and pleural lining as well as pleural plaques and effusions. Non-smoking individuals with asbestosis usually develop small airway restrictive disease, and those who smoke typically develop an obstructive, restrictive pattern of lung disease. Inhaled asbestos fibers can produce mutagenesis and carcinogenesis in the pleural lining and lung causing pleural mesothelioma or adenocarcinoma.

d. Emergency Treatment

Treatment for asbestos exposure is supportive in nature. There is no antidote. Patients with chronic symptoms should avoid smoking (ATSDR, 1995b; 1990b; Leikin & Paloucek, 1998).

Benzene

a. Description

Benzene, the simplest member of the aromatic series of hydrocarbons, is a clear, colorless highly volatile liquid with a sweet odor. Sources include natural gas and crude oil products of petroleum and the petrochemical industry. It is immiscible with water and dissolves fats. It undergoes oxidative degradation to form phenol, pyrocatechol, and mucic acid. The benzene alkyl derivatives are oxidized to benzoic acid. The major routes for benzene absorption are by inhalation, ingestion, and dermal contact. The Occupational Safety and Health Administration recommendation for maximum benzene contaminants is one part per

million (1 ppm) in ambient air. The U.S. Environmental Protection Agency (EPA) recommends five parts per million (5 ppm) in drinking water as the maximum contaminant. Benzene is metabolized in the liver and bone marrow.

b. Uses

Benzene is used commercially in rubber fabrication, high-speed printing processes, paint manufacturing, and in the plastic industry. It is also used as an additive to gasoline for anti-knock purposes, as a solvent in chemical laboratories, and as a component of industrial cleaning agents.

c. Signs and Symptoms

Symptoms of benzene exposure include burning sensation of the skin, eyes, respiratory tract, mouth, and stomach, as well as chest pains, cough, headache, giddiness, vertigo, ataxia, mydriasis, paresthesia, ototoxicity, tinnitus, confusion, stupor, and coma.

Effects on the hematopoietic system from chronic exposure to high levels of benzene can result in a decrease in erythrocytes, and the occurrence of leukopenia, thrombocytopenia, aplastic anemia, and leukemia. Death is caused by respiratory failure secondary to respiratory inflammation and lung hemorrhages, CNS depression or ventricular fibrillation.

d. Emergency Treatment

There is no antidote for benzene. Secondary contamination of rescue personnel can occur. Assistance from a local HAZMAT (Hazardous Material) Team or trained personnel is required. The HAZMAT team or other trained personnel will remove the patient from the source of exposure, remove contaminated clothing, and continuously rinse exposed skin and eyes with copious amounts of water. In cases of ingestion, do not induce emesis. Activated charcoal, 100% humidified oxygen, and parenteral fluids may be administered if necessary. Convulsions may be controlled with diazepam. Epinephrine should be avoided to prevent inducing ventricular fibrillation (Leikin & Paloucek, 1998; ATSDR, 1994a).

Carbon Disulfide

a. Description

Carbon disulfide is a clear, colorless, highly flammable, volatile liquid. It is very stable in water. In its pure state the odor is like that of ether or chloroform. The impure commercial grade of carbon disulfide smells like rotten eggs or decaying cabbage. Carbon disulfide can explode in the air and catches fire very easily.

Primary entry of carbon disulfide is by inhalation, ingestion of water or foods that contain it, and skin absorption. Rayon plant workers are among those at highest risk for exposure. Communities located near industrial sites releasing carbon disulfide emissions are also at risk of exposure. NIOSH recommends an exposure limit for workers of 1 ppm in air.

b. Uses

Carbon disulfide is used as a solvent in the production of artificial fibers, particularly rayon, flotation agents, rubber industry, waxes and resins. Carbon disulfide is used in the production of cellophane and semi-conductors, matches, instant color photography, corrosion inhibitors, and gold and nickel plating. The vapor has been used as a disinfectant, insecticide, and pesticide. It is an essential

ingredient in the manufacture of carbon tetrachloride.

c. Signs and Symptoms

Carbon disulfide inhibits cellular function and damages liver enzyme systems. Exposure to 60 to 100 ppm for a short time can result in severe intoxication and death. Exposure to 5,000 ppm is rapidly fatal.

Manifestations of acute toxicity include extreme irritability, uncontrolled anger, rapid mood changes including manic delirium, hallucinations, paranoid ideas and suicidal tendencies. Other symptoms include mild to moderate irritation of skin, eyes, and mucous membranes caused by percutaneous absorption; headache, garlic-smelling breath, nausea, vomiting and diarrhea, occasional abdominal pain, weak pulse, palpitations, fatigue, weakness in legs, unsteady gait, tremors, vertigo, hallucinations, nystagmus, diplopia, seizures, psychosis, and CNS depression with respiratory paralysis. Death may occur following a convulsion, coma or respiratory paralysis.

Manifestations of chronic toxicity include tremors, weakness, paralysis, peripheral neuritis, absent corneal reflex, hypertension, atherosclerosis, renal and parenchymal lesions, fatigue, loss of memory, feelings of inadequacy, frank psychosis, excessive sleep, and short-term memory disturbances.

d. Emergency Treatment

There is no antidote for carbon disulfide exposure. The victim should be removed from source of exposure, contaminated clothing removed and exposed skin and eyes rinsed with copious amounts of water. The victim should be transported to the emergency room where emesis will be instituted. Diazepam may be given for convulsions. Recovery from exposure to carbon disulfide usually occurs within a few months to a year. However, any paralysis that occurs will be permanent (Leikin & Paloucek, 1998; ATSDR, 1992b).

Carbon Monoxide

a. Description

Carbon monoxide is a clear, colorless, odorless, tasteless gas formed by inefficient or incomplete combustion and/or oxidation. It is found in the exhaust from all internal combustion engines, as well as anything that burns oil, gas or wood. Each year many people are killed because of fumes released from improperly vented furnaces, water heaters, wood stoves, space heaters, and fireplaces.

b. Uses

There are no commercial uses for carbon monoxide.

c. Signs and Symptoms

The pathology of carbon monoxide poisoning consists of high concentrations of carboxyhemoglobin in the circulating erythrocytes leading to tissue hypoxia and inhibition of cellular respiration. Manifestations of carbon monoxide poisoning include bright red skin color (cherry red), syncope, hypoxia, frontal headache, dizziness, sinus bradycardia and tachycardia, dyspnea with hyperventilation, tinnitus, hypothermia, short-term memory deficit, angina, weakness, nausea, dimness of vision, disorientation, lethargy, unconsciousness, coma, seizures, and death from respiratory arrest.

d. Emergency Treatment

Emergency treatment includes removal of the patient from the exposed area and assessment of the need for artificial respiration. The victim should be transported to an emergency room for delivery of 100% oxygen by mask. The antidote for carbon monoxide poisoning is hyperbaric oxygen therapy, which displaces carbon monoxide from binding sites and decreases cerebral edema. Hyperbaric oxygen therapy is used for exposed patients who are pregnant, have acute neurotoxicity or angina with a carboxyhemoglobin level of 20% (Leikin & Paloucek, 1998).

Chlordane (contains Heptachlor, Aldrin, Dieldrin)

a. Description

Chlordane is a thick, liquid, polycyclic, chlorinated hydrocarbon whose color ranges from colorless to amber. It has a mild, irritating smell and low water solubility. Exposure to chlordane, an organochlorine, produces a cumulative and neurotoxic effect by interfering with sodium and potassium movement across cell membranes. Chlordane is biotransformed by hepatic microsomal enzymes, stored in adipose tissue, and excreted in urine.

Absorption by humans depends on the presence of contaminated soil adhered to organic matter (This sentence seems to apply more to the last sentence in this paragraph - suggest moving or explaining more here.). Once absorbed, chlordane does not desorb. It does not leach or diffuse into the hydrosphere from contaminated soil. Dispersion of chlordane is through soil particles, which remain biologically active for more than 30 years. Chlordane exposure occurs mainly by indoor inhalation of the volatilized fumes from prior application on wood or ground surfaces. Dermal exposure from soil as well as ingestion and eye contact may also occur.

b. Uses

Until prohibited in 1988, chlordane was used as a termiticide and insecticide. Supplies of chlordane may still exist in warehouses, garages, and landfills.

c. Signs and Symptoms

Chlordane causes gastrointestinal and CNS damage. Acute chlordane poisoning disrupts nerve transmissions resulting in CNS excitation, convulsions, tachycardia, and respiratory depression. Signs and symptoms include irritability, Reye's-like syndrome, hyperexcitability, hyperreflexia, tremors, convulsions, and periods of depression.

Chronic exposure can cause headaches, light headedness, tremors, weakness, scleroderma, seizures, liver damage, and, possibly, liver cancer.

d. Emergency Treatment

Emergency treatment requires properly trained rescuers. Secondary exposure to chlordane can occur. There is no antidote for chlordane. The victim should be assessed for patent airway and breathing. Victims who are able may assist with removal of contaminated clothing. Exposed eyes and skin require washing with copious amounts of water. In case of chlordane ingestion, do not induce emesis in patients with seizure activity because of possible aspiration. Activated charcoal may be given to a conscious patient. Saline cathartics or cholestyramine may be given to help bind

and eliminate chlordane. The patient must avoid eating fats, oils, and demulcents. Do not give epinephrine, other adrenergic drugs or atropine because enhanced myocardial irritability occurs and predisposes the patient to ventricular fibrillation (EPA, 1989; Leikin & Paloucek, 1998; ATSDR, 1994a; ATSDR, 1995a).

Chloroform (Trichloromethane)

a. Description

Chloroform is a heavy, clear, colorless liquid with an ether-like odor. It is formed by the action of chlorinated lime on methyl alcohol. Chloroform is biotransformed to an active metabolite (phosgene gas) that covalently binds to hepatic proteins. Exposure occurs by inhalation, skin/eye contact, and by ingestion.

b. Uses

Chloroform is used as a solvent, grain fumigant, tincture, anesthetic agent, and aerosol propellant.

c. Signs and Symptoms

Signs and symptoms of acute toxicity include burning eyes, corneal irritation, nausea and vomiting, jaundice, mydriasis, nystagmus, acetone breath, depression, coma, cardiac arrhythmias, pneumonitis, pulmonary edema, and respiratory depression. Chloroform also causes liver and kidney cell damage.

Chronic exposure can cause brain changes and psychotic behavior, dizziness, hemolysis, and hepatitis.

d. Emergency Treatment

Emergency treatment for exposure to chloroform includes removing the patient from the area of exposure and removing contaminated clothing. The victim's exposed skin should be washed thoroughly with copious amounts of water. After the patient is transferred to the emergency room, emesis will be instituted within 30 minutes or lavage and charcoal instillation within 60 minutes. Supportive therapy for treatment of cardiac and respiratory problems should be initiated. Atropine sulfate may be administered for severe bradycardia to block muscarinic receptors and reduce cardiac depression (Leikin & Paloucek, 1998).

DDT (Dichlorodiphenyltrichloroethane or 1,1,1-Trichloro-2,2-bis(*p*-chlorophenyl) ethane)

a. Description

DDT is an organochlorine pesticide and member of a widely used group of compounds that causes excitation or depression. The substances and their metabolites accumulate in the natural food chain. Organochlorines tend to accumulate in the body.

Ingestion, inhalation, and dermal absorption are routes of exposure for DDT. Human exposure occurs through the food chain and breast milk. Exposure also occurs through herbicide, chemical, and waste handling.

b. Uses

Historically DDT was used as a pesticide on cotton, peanut, and soybean plants and to kill insects that carry malaria and typhus. Because of the danger to wildlife and humans, the use of DDT has been banned in the United States since 1972. However, DDT is still

manufactured and exported by the United States. Exposure may be dermal, inhalation or oral.

c. Signs and Symptoms

The mechanism of DDT toxicity is probably its interference with sodium and potassium transfer across cell membranes. The half-life of the chemical in humans is seven years.

Signs and symptoms of poisoning from DDT are gait disturbance, dizziness, malaise, fatigue, headache, nausea, vomiting, tremors, optic neuropathy and paresthesia of mouth and lower part of face. Exposure to a large amount of DDT can result in excitability, tremors, and convulsions (Andrews, 1992).

DDT is listed as a probable human carcinogen. Liver cancer has been found in animals that were fed grain containing DDT. Breast cancer may be associated with chronic DDT exposure (Leikin & Paloucek, 1998).

d. Emergency Treatment

Wash the exposed area with soap and copious water. Irrigate exposed eyes with water. For oral ingestion, do not induce emesis. Lavage may be performed within one hour. Multiple doses of charcoal and cholestyramine will enhance elimination (Andrews, 1992; ATSDR, 1994c; Leikin & Paloucek, 1998).

Dioxin (2,3,7,8- Tetrachlorodibenzo-*p*-dioxin, TCDD)

a. Description

Dioxin (TCDD) was a contaminant of chlorophenoxy herbicides and the most toxic of the 75 dioxin congeners. TCDD has been associated with increased risk of cancer. Dioxins accumulate in the food chain and in body fat. Certain industrial processes, such as the manufacture of chlorinated herbicides, germicides, and organic solvents, may expose workers to the contaminant during industrial manufacturing processes. Transformer or capacitor fires and hazardous waste site fires also expose workers to dioxin. Routes of exposure include dermal exposure, inhalation, and ingestion of contaminated meat, milk, and fish.

b. Uses

Dioxins have no known use but are unintended byproducts of several chemical processes. TCDD was a contaminant in chlorophenoxy herbicides used to control the growth of bushes and in the defoliant Agent Orange used during the Vietnam war. It is no longer manufactured in this country. Mixtures of dioxins have been found in the combustion of fossil fuels, wood, and during waste incineration.

c. Signs and Symptoms

One mechanism of dioxin toxicity is the inhibition of folate synthesis. Symptoms of dioxin toxicity include gastroenteritis, skeletal muscle myotonia, myoglobinuria, cardiac dysrhythmia, CNS depression, and chloracne (comedones, cysts, pustules, and abscesses). Hepatic dysfunction, peripheral neuropathy, fat metabolism disorders, and elevated serum cholesterol levels are common findings associated with industrial exposure.

Chronic exposure to TCDD has been associated with soft tissue sarcoma, Hodgkin's disease, non-Hodgkin's lymphoma, and gastric cancer.

d. Emergency Treatment

Emergency treatment for acutely exposed persons includes removing the person from the source of contamination. Assess victim for patent airway, breathing, and circulation. Decontamination procedures should be performed by specially trained personnel (Bronstein & Sullivan, 1992; ATSDR, 1990c).

Chronically exposed individuals will require a complete medical workup and toxicological profile. Hepatic transaminase levels should be monitored (Haddad et al., 1998; Leikin & Paloucek, 1998).

Formaldehyde (Formalin)

a. Description

Formaldehyde is a colorless gas with a pungent odor made by the oxidation of methyl alcohol. It is highly toxic and flammable. Formaldehyde occurs naturally in the environment. It is also found in minute amounts in the human body. It is very soluble in water. Exposure occurs through inhalation, skin/eye contact, and ingestion. Formaldehyde may be carcinogenic.

b. Uses

Formaldehyde is used in some building insulation and for fireproofing. It is also used as a disinfectant, fumigant and germicide, and embalming fluid, and in the manufacture of glues, lacquers, tannery, and textile products.

c. Signs and symptoms

Signs and symptoms of formaldehyde poisoning are dyspnea, cough, epigastric pain, nausea, vomiting, vertigo, urticaria, drowsiness, ataxia, anxiety, tachycardia, renal failure, coma, and collapse. Inhaled formaldehyde is irritating to mucous membrane of the nose, upper respiratory tract, and the eyes. Inhalation can cause hypersensitivity reactions. Because odor adaptation can occur it is not a reliable warning of the presence of formaldehyde. Ingested formaldehyde causes inflammation and ulceration of the gastrointestinal tract, gastritis, circulatory collapse, renal failure and acidosis. Toxicity causes cellular necrosis. Death is due to respiratory failure.

The major concerns of repeated formaldehyde exposure are sensitization and cancer. In sensitized persons, formaldehyde can cause asthma and contact dermatitis. In persons who are not sensitized, prolonged inhalation of formaldehyde at low levels is unlikely to result in chronic pulmonary injury.

d. Emergency Treatment

Victims with contaminated clothing or skin can cause secondary exposure to rescue workers by direct contact or through off-gassing of formaldehyde vapors. Emergency treatment requires special training or consultation with a regional HAZMAT team. Rescue workers will remove victim's clothing and flush skin or eye with copious water prior to transport to an emergency room. Vomiting should not be induced. Have the victim drink water to dilute the contents of the stomach .

There is no antidote for formaldehyde toxicity. Treatment by trained personnel consists of treatment for shock, morphine for pain, antibiotics, and sodium bicarbonate for acidosis. Emetics are not to be given and gastric lavage is not to be initiated. Sodium bicarbonate may be given to neutralize hydrogen concentration and raises blood pH (Leikin & Paloucek, 1998; ATSDR, 1994a).

Glycol Ethers (Ethylene Glycol Monomethyl Ether)

a. Description

Glycol ethers are alkyl derivatives of ethylene and trimethylene glycol, which represent a group of widely used solvents. Glycol ethers are applied in surface coatings, fingernail polishes and removers, dyes, writing inks, cleaners, and degreasers. There is significant exposure by inhalation and dermal exposure. Liquid glycol ethers are readily absorbed through the skin but their vapors are not. Exposure through ingestion can also occur.

b. Uses

There are various uses for each form of the glycol ethers (EGME, EGEE, and EGBE) including use as insecticides and resins, in jet fuel and wood stains, and in production of microelectronics, and antifreeze agents.

c. Signs and Symptoms

Acute exposure to these solvents produces three phases of effects: CNS depression, cardiovascular dysfunction within 12 hours, and proteinuria and acute renal failure within 12 to 72 hours. Other symptoms are drowsiness and irritation of the mucous membranes, eyes, and respiratory tract; hyperventilation; hypotension; headache; confusion; and tachycardia. Symptoms may be delayed for 18 hours. Harmful effects include metabolic acidosis, hypocalcemia, and renal toxicity.

Chronic exposure is associated with central or peripheral neurotoxicity. Ingestion can result in severe metabolic acidosis, leading to severe acute nephrotoxicity. The kidney is the primary target organ. Glycol ethers have been associated with birth defects and other reproductive problems.

d. Emergency Treatment

Emergency treatment of acute eye exposures includes flushing the affected area with large amounts of water at low pressure for five to ten minutes while awaiting transport to the emergency room. Emesis will be instituted for ingestion exposures within 30 minutes of exposure. Lavage and charcoal instillation will be initiated within 60 minutes of exposure. Sodium bicarbonate may be give for acidosis (Leikin & Paloucek, 1998).

Chronic exposure resulting in toxic levels of glycol ethers may require aggressive management of acidosis, hemodialysis and administration of ethanol (Spyker & Sullivan, 1992).

Lead

a. Description

Lead may be found in buildings, air, food, industrial dust, leaded gasoline, lead-glazed pottery, and some folk remedies. Lead is easily absorbed as a fume or as particulate matter. Lead is not absorbed readily in the adult gastrointestinal tract but is absorbed easily in the intestinal tract of children. Once absorbed, lead binds to erythrocytes and is distributed throughout the body in the bones, teeth, liver, lung, kidney, brain, and spleen. Lead accumulates and is stored in bone tissue. It may be later released from bone causing further toxicity. Pregnant women with lead exposure may transfer lead along with calcium to the developing fetus via the placenta. Small amounts of lead are excreted by the kidneys and by the intestinal tract.

Absorbed lead is harmful to the enzyme system involved in heme synthesis resulting in anemia. Renal effects of lead intoxication include hypertension and renal failure. Reproductive effects because of occupational exposure to lead are decreased fertility, spontaneous abortions, stillbirth, and increased infant mortality (Keogh, 1992).

The principal exposure of children to lead occurs through hand-to-mouth ingestion of lead-based paint chips, ingestion of contaminated water and soil, and inhalation of lead dust in air. Homes built before 1978 may contain lead-based paint in the interior and exterior . Children who live in housing built before 1950 are at the greatest risk of exposure because of the high level of lead in paint used on that era. Other exposure sources are imported ceramic ware, already stated above, soil or dust near lead industries, and many hobbies such as stained glass art works.

Lead intoxication causes damage to the central and peripheral nervous systems, blood-forming organs, and gastrointestinal tract. Lead concentrates in the gray matter of the brain and produces cognitive and motor deficits. It also produces peripheral neuropathy. Untreated excessive lead levels in children cause encephalopathy producing permanent brain damage and retardation. The OSHA permissible exposure level of lead in the workplace is 50 $\mu g/m^3$ as an 8 hour time-weighted-average.

b. Uses

Lead has many commercial uses including the smelting process, soldering, cable cutting and splicing, auto repair, radiator repair, welding, grinding, battery manufacturing, printing, cosmetics, ceramics, folk remedies, and plumbing. Lead has been prohibited in household paint since 1977.

c. Signs and Symptoms

Signs and symptoms of mild lead toxicity include myalgia, fatigue, irritability, and abdominal discomfort. Moderate toxicity symptoms include burning pain in the stomach, anorexia, headache, nausea, weight loss, vomiting, constipation, arthralgia, general fatigue, and deafness. Severe toxicity symptoms include muscle weakness, tremor, seizures, palsies, blindness, and encephalopathy leading to stupor, convulsions or coma.

d. Emergency Treatment

Treatment for lead poisoning consists of reducing/removing the individual from the exposure and maintenance of fluid and electrolyte balance. Once the individual has been removed from further lead exposure, chelation therapy may be started . Chelation therapy consists of drugs designed to remove lead from storage depots in the body. The chief antidote for lead poisoning is parenteral BAL (dimercaprol) and calcium disodium ethylenediaminetetraacetic acid (EDTA). Recently, oral dimercaptosuccinic acid (DMSA, Chemet, Succimer) has been used successfully. Therapy should be continued for three to six months for children and two months for adults as needed. D-penicillamine is an oral drug that increases the excretion of lead in the urine. Penicillamine is indicated for patients unable to take BAL, EDTA or Succimer. The patient should be monitored for blood lead levels, erythematous rashes, hypersensitivity reactions, anemia, hypercalcemia, fever, neutropenia, thrombocytopenia, eosinophilia, proteinuria, and kidney damage (Keogh, 1992;

ATSDR, 1992a; Green, 1997; Leikin & Paloucek, 1998).

Manganese (Manganese Dioxide)

a. Description

Manganese is a gray, hard, brittle metallic element that decomposes in water. Manganese dioxide is a black crystalline solid or powder that is insoluble in water. In humans, manganese is an essential trace element necessary for metabolic activity in cells. Compounds of manganese may be absorbed by ingestion, inhalation or skin contact. Manganese exposure can occur in the mining, smelting or refining of manganese ores, near crushing operations and furnaces that produce alloys and steel, or in the manufacture of manganese welding rods. The half-life of manganese is 30 hours. Manganese catalyzes dopamine depletion and free radical production within the CNS resulting in cortical and cerebellar degeneration.

b. Uses

Manganese is used in fertilizer for grapes and tobacco, welding rods, ceramics, electrical coils, matches, glass, animal food additives, paints, rubber, preservatives, pesticides, dry cell batteries, as an oxidizing agent in chemical industry, as an anti-knock agent in unleaded gasoline, and in jet fuel.

c. Signs and Symptoms

Exposure to manganese fumes produces flu-like symptoms with fever, chills, and body aches , as well as cough, pleuritis, bronchitis, asthma, pneumonitis, gastrointestinal irritation, and pancreatitis.

The major chronic health effect is a neurologic syndrome is called “manganism,” manifested by anorexia, asthenia, apathy, somnolence, headaches and decreased social interaction. Chronic exposure produces fatigue, headache, apathy, behavioral disturbances, excitability, tremor, bradykinesia, gait disturbance, masked facies, excessive salivation, vasomotor disturbance, and liver enzyme elevation.

d. Emergency Treatment

Emergency treatment includes removing the victim from the source of exposure while awaiting transport to the emergency room. Breathing and a patent airway should be maintained. Dermal exposures should be washed with soap and rinsed promptly with large amounts of water. Eye exposures should be rinsed with large amounts of tepid water. Trained personnel should handle acute ingestion by inducing vomiting or gastric lavage (Leikin & Paloucek, 1998).

Chronic symptoms of Parkinsonism are treated with levodopa and 5- hydroxytryptophan drug therapy (Haddad et al., 1998).

Mercury (Methylmercury, Elemental Mercury, and Inorganic Mercury)

a. Description

Elemental mercury is a silver-gray liquid at room temperature. Elemental mercury is the source of most occupational exposure. Inorganic mercury (mercurous chloride)is used to inhibit bacterial or fungal growth. Organic mercury (methyl mercury) is formed by bacteria from off-gassing of mercury from soils and surface waters and the disposal of solid waste containing mercury in landfills. Contamination of water results from the weathering of mercury

in certain rocks and industrial run off.

The absorption and metabolism of mercury varies with its chemical and physical form. Elemental mercury is almost completely absorbed through inhalation. It diffuses rapidly across placental and blood-brain barriers. It is poorly absorbed through the gastrointestinal tract. The biologic half-life of elemental mercury is 60 days and is excreted in the urine and feces. Inorganic mercury or mercury salt does not cross the blood-brain barrier and has a half-life of 40 days. Organic mercury is absorbed well by inhalation, dermal contact, and ingestion. Organic mercury is teratogenic. The biologic half-life is 70 days.

Mercury ions alter the structure and function of enzymes and other proteins by binding to sulfhydryl groups. It interferes with cellular metabolism by binding phosphoryl and amine groups. Elemental mercury vapor and organic mercury exposure can have serious adverse effects on the neurologic status of a fetus and adults. Mercury can easily accumulate in fish found in contaminated streams and rivers.

b. Uses

Mercury is used to prevent bacterial and fungal growth in paint manufacture. It is also used in laboratories; in the manufacture of fireworks, amalgams, and button batteries; and by dye makers, fur processors, photographers, electroplaters, taxidermists, jewelers, and embalmers. Mercury has multiple uses in folk remedies. Metallic mercury is used by Mexican-Americans and Asian populations for stomachaches. Some Latin American and Caribbean populations use mercury in religious rituals.
Metallic mercury and its vapors are extremely difficult to remove from such items as clothes, furniture, carpet, floors, and walls. The contamination can remain for months or years, posing a risk to exposed individuals.

c. Signs and Symptoms

Early symptoms of mercury neurotoxicity are nonspecific and include malaise, blurred vision, hearing loss, ataxia, dysarthria, and paresthesias. Ingestion of inorganic mercury compounds can result in gastrointestinal bleeding due to necrotizing ulcer of the GI tract and shock. Accumulation of mercury in the kidneys can produce a nephrotic syndrome affecting the permeability of tubular epithelium. Acute tubular necrosis and renal failure may follow. Severe lung tissue damage, pulmonary edema, alopecia, loss of memory, stomatitis, and tremor have occurred with elemental mercury poisoning.

Chronic exposure to any form of mercury primarily affects the nervous system. Inorganic mercury can also affect the kidneys.

d. Emergency Treatment

Treatment of inhaled elemental mercury and inorganic mercury poisoning usually involves the use of chelation therapy with dimercaprol. Treatment for organic mercury compounds includes oral D-penicillamine to enhance elimination. The antidote for inorganic mercury poisoning is oral DMSA (Succimer). Ingestion of mercury salts (inorganic mercury) should be treated by trained personnel with oral lavage within one hour followed by charcoal instillation. Emesis should not be used for inorganic mercury ingestion because this type of mercury is corrosive in nature. Administration of intravenous fluids helps to reduce the concentration of mercury in the kidneys and promotes elimination. Hemodialysis may be

employed if necessary. BAL is not recommended for elemental or organic mercury poisoning because it may cause redistribution of mercury to the brain (ATSDR, 1992d, 1997; Leikin & Paloucek, 1998).

Methanol (Wood Alcohol, Wood Naptha)

a. Description

Methanol is a colorless, inflammable, volatile liquid with a faintly pleasant odor. Exposure to methanol occurs mainly through ingestion (accidental ingestion through siphoning of gasoline) and by skin and inhalation absorption. Methanol is slowly metabolized to formaldehyde and rapidly converted to formic acid. Formic acid interferes with cellular respiration.

Ingestion of methanol produces an intoxication consisting of an initial mild inebriation. Metabolic acidosis occurs 24 hours after ingestion and is fatal if untreated. Prior to acidosis, partial or complete blindness may occur due to atrophy of the ganglion cells of the retina. Death can occur because of respiratory failure.

b. Uses

Methanol is used as a solvent for paints, varnishes, and paint removal. However, illicit use has been made of methanol either accidentally or deliberately as a substitute for ethyl alcohol.

c. Signs and Symptoms

Signs and symptoms of methanol toxicity include inebriation, headache, muscle weakness, nausea, vomiting, back pain, abdominal pain, delirium, blurred vision or diplopia, neuropathy, Parkinson-like syndrome, mild dementia, cyanosis, severe acidosis, renal failure, and coma.

d. Emergency Treatment

Emergency treatment includes inducing emesis if the victim is conscious or gastric lavage if the person is not alert. The antidote for acidosis is intravenous sodium bicarbonate. The antidote for formic acid formation is intravenous ethanol to decrease formation of lethal metabolites. Hemodialysis may be employed for renal failure. Additional treatment includes intravenous folic acid or leucovorin. Treatment should begin within two hours of ingestion to prevent ocular toxicity. Treatment for shock and oxygen therapy may be necessary. The eyes should be protected from light (ATSDR, 1992c; Blain, 1994).

Methylene Chloride (Dichloromethane, Methylene Bichloride, Methylene Dichloride, Methane Dichloride)

a. Description

Methylene chloride is a clear, colorless liquid with a pleasant odor. It is volatile and produces potentially toxic concentrations at room temperature. It is slightly soluble in water and most organic solvents. It is used in food, furniture, and plastics processing; as a paint remover; as a cleaning agent for electronic boards and metal parts; and as a propellant in hair sprays and room air fresheners. More than 90% of the methylene chloride released to the environment changes to carbon dioxide. The chief routes of exposure are inhalation, ingestion, and dermal contact. Methylene chloride is absorbed easily into the blood stream after inhalation and ingestion. Inhalation is the major route of exposure. Vapors of

methylene chloride are heavier than air and may cause asphyxiation. Vapors can cause skin and eye chemical burns.

b. Uses

Methylene chloride is an important solvent in paint, degreasing agents, photographic film, synthetic fibers, pharmaceuticals, adhesives, inks, and printed circuit boards. It is used to extract caffeine and fat from drugs, coffee, and food.

c. Signs and Symptoms

Symptoms of acute exposure to methylene chloride are CNS depression, loss of consciousness, seizures, coma, respiratory irritation, cardiac dysrhythmias with decreased heart contractility, increased pulse rate, accumulation of fluid in the lungs, liver dysfunction, skin irritation, and inflammation of eye surface and iris. Rapid loss of consciousness and death have been reported.

Chronic exposure can raise levels of carboxyhemoglobin, damage the liver, and in laboratory animals cause cancer.

d. Emergency Treatment

Emergency treatment requires a HAZMAT team to decontaminate the victim. Patency of airway and breathing status should be assessed. Contaminated clothing should be removed. Contaminated skin and hair should be rinsed for two to three minutes, washed with mild soap, and rinsed thoroughly. Exposed or irritated eyes should be flushed with plain or saline water for three to five minutes. In cases of ingestion, emesis should not be induced. Activated charcoal should be administered if the victim is alert (ATSDR, 1994a).

Methyl Ethyl Ketone (2-Butanone)

a. Description

Methyl ethyl ketone is a colorless, flammable liquid. It occurs naturally in certain foods and beverages. It evaporates when exposed to air and dissolves in water. Exposure routes for methyl ethyl ketone are inhalation, dermal exposure, and ingestion. It is distributed widely in tissues and may cross the placenta and enter the human fetus. The plasma half-life is 49 to 96 minutes.

Exposure to methyl ethyl ketone can occur in the workplace or in the environment following its release into air, land, or groundwater. Exposure can occur through contact with products containing small amounts of methyl ethyl ketone, from contaminated air, food or water; or from dermal contact.

b. Uses

Methyl ethyl ketone is used as an additive to protective coatings, adhesives, printing inks, paint removers, and special lubricating oils. It is used to make synthetic leather, aluminum foil, other chemicals, drugs of abuse, and cosmetics. Small amounts are used to sterilize surgical instruments, hypodermic needles, and dental instruments.

c. Signs and Symptoms

Inhalation of methyl ethyl ketone causes headaches, dizziness, nausea, numbness in fingers and toes, panic attacks, hypotension, tachycardia, conjunctival irritation, mydriasis, and unconsciousness. The vapor is irritating to the eyes, nose, and throat.

There is no data on the effects of chronic

exposure to methyl ethyl ketone.

d. Emergency Treatment

The victim should be removed from the source of contamination while awaiting transport to the emergency room. Breathing and patent airway should be maintained. For skin contact, the affected area should be washed with soap and rinsed with copious amounts of water. Contaminated clothing should be removed and isolated. For eye exposure, contact lenses should be removed and the victim's eyes should be flushed with copious amounts of water. Do not put anything in the eyes such as ointments or other medicines. For ingestion exposure, do not induce vomiting because of the possibility of aspiration. Lavage with a cuffed endotracheal tube in place. Activated charcoal may be instilled (U. S. Environmental Protection Agency, 1994; Leikin & Paloucek, 1998).

Parathion and Methyl Parathion, Alkeron (Alleran, Danthion, DNTP, DPP)

a. Description

Parathion and methyl parathion are organophosphates that are highly toxic. In a concentrated state, they are a brownish liquid with an odor of garlic. When diluted with water, methyl parathion turns milky white. It is usually dissolved in hydrocarbon solvents such as toluene or xylene, which are flammable. It leaves a yellow stain on areas where it has been sprayed. Open air, sunlight, and rain break down methyl parathion into harmless byproducts.

Routes of exposure are dermal and mucous membrane exposure, inhalation following spraying, and by ingestion of contaminated foods and water. Parathion, as all organophosphate pesticides, alters the cholinergic synaptic transmission at neuroeffector junctions, myoneural junctions, autonomic ganglia, and in the CNS.

b. Uses

Parathion is used as a pesticide on cotton, soy beans and vegetable fields in the southern part of the United States. It should never be used in homes or buildings as it does not decompose quickly in indoor areas. Methyl parathion has been used illegally as a pesticide to control roaches and pests in homes, businesses, day care centers, and other establishments in parts of southern Mississippi, Tennessee and Arkansas.

c. Signs and Symptoms

In mild to moderate parathion and methyl parathion poisoning, the symptoms are non-specific: headaches, nausea, vomiting, diarrhea, and dizziness. Signs and symptoms of severe poisoning include salivation, pinpoint pupils, blurred vision, bradycardia, muscle fasciculation, diarrhea, irritability, and lethargy. A diagnosis can be made through blood testing of red cell cholinesterase inhibition (preferred) and urine testing for p-nitrophenol (P-NP), a specific metabolite of methyl parathion, within 24 hours of suspected exposure.

d. Emergency Treatment

For minor exposures, such as breathing the solvent, the victim should be removed to fresh air. Serious poisoning will require the assistance of the HAZMAT team. Rescue workers will wash exposed areas with mild soap and rinse thoroughly with copious amounts of water. All contaminated clothing

should be removed and double-bagged prior to transporting the victim to the emergency room.

In cases of ingestion, emesis should not be induced but activated charcoal may be administered. The antidote for parathion poisoning is atropine sulfate. In addition pralidoxime (2-PAM, Protopam) is indicated for seriously poisoned victims (ATSDR, 1994a).

Petroleum Distillate (Naphtha, Stoddard Solvent)

a. Description

Petroleum distillate is a colorless, flammable liquid with an odor similar to kerosene or gasoline. Exposure to petroleum distillate occurs through inhalation or dermal contact with dry-cleaning products, paints, paint thinners, lacquers, coal tar, rubber cement, furniture refinishing products or residues on dry-cleaned products. Exhaust from dry-cleaning plants and contamination of water and soil from waste landfill discharges are other sources of petroleum distillate exposure.

b. Uses

Petroleum distillate is a multipurpose solvent for dry cleaning.

c. Signs and Symptoms

Acute exposure to petroleum distillate causes CNS excitation followed by CNS depression, headaches, memory deficits, dizziness, nausea, vomiting, fever, tachypnea, erythema, and inebriation. High concentration exposure can irritate the respiratory tract: coughing, choking, and respiratory rales. Acute ingestion and aspiration can produce a chemical pneumonitis and acute respiratory distress, pulmonary edema, and emphysema. Dermal exposure produces dermatitis and vesicular lesions.

There is little data on the effects of chronic exposure.

d. Emergency Treatment

Emergency treatment of petroleum distillate exposure includes removing the person from the source of exposure and removing contaminated clothing while awaiting transport of the victim to the emergency room. Exposed areas should be washed with soap and water. Eye splashes should be irrigated with saline or water for at least 15 minutes or until pain resolves. Patent airway and breathing should be maintained. Emesis should not be induced. Because of the risk of aspiration, emesis and gastric lavage should not be performed (ATSDR, 1993b; Leikin & Paloucek, 1998).

Polychlorinated Biphenyls (PCBs)

a. Description

Polychlorinated biphenyls (PCBs) are a group of industrial chemicals that share a common structure. There are over 200 compounds (congeners) that have harmful effects on humans. PCBs are oily liquids or solids that are colorless or light yellow in color. PCBs have no smell or taste. They are good electrical insulators and are nonflammable. The manufacture of PCBs has been banned in this country since 1977. PCBs enter the environment from accidental spills or from leaks or fires in pre-1977 transformers or capacitors containing PCBs. Illegal dumping of PCBs in landfills that are not specially equipped and burning of organic wastes account for further releases into the environment. PCBs remain in the air for 10 days and are transported elsewhere by wind, snow, and rain. PCBs can be found in water years after the initial contamination occurred and can accumulate in fish.

The sources of exposure are the environment and the workplace. People who live near hazardous waste sites are exposed to PCBs in the air. Children are exposed by touching and eating contaminated soil near waste sites. Infants are exposed to PCBs in utero and while breast feeding from exposed mothers. Eating fish, seafood, dairy products, or fatty meats contaminated with PCBs is another source of exposure.

PCBs are absorbed rapidly but are metabolized and excreted slowly. Excretion of PCBs is mainly in bile; metabolites are excreted in the urine. The half-life of PCBs in rats ranges from one to 460 days, depending on the degree of chlorination in the PCB mixture. There are essentially no pharmacokinetic data for humans.

b. Uses

PCBs were used as coolants, in capacitors and transformers, insulating materials, and lubricants for gas turbine engines and may be found in fluorescent lighting, old microscope oil, and hydraulic fluids.

c. Signs and Symptoms

Signs and symptoms of acute exposure include chloracne, which appears weeks or months after exposure, headache, fatigue, nausea, vomiting, hepatitis, pruritus, elevated liver enzymes, hepatomegaly, and hyperpigmentation of skin and nails.

Symptoms of chronic exposure may be absent or may include weight loss, anorexia, nausea, vomiting, jaundice, and abdominal pain. PCBs affect the skin and liver and may have developmental effects. PCBs are considered to be probable carcinogens.

d. Emergency Treatment

Emergency treatment of acute exposure includes removal from the source of exposure, removal of contaminated clothing, irrigation of eye splashes with tepid water for at least 15 minutes followed by ophthalmic examination. Ingestion of PCBs should be treated by trained personnel by instituting gastric lavage within one hour and instillation of activated charcoal. Exposed persons should have periodic follow-up examinations with particular attention to hepatic function and dermal lesions. There is no antidote for PCB exposure.

Treatment of chronic exposure is symptomatic and includes warning the person to avoid exposure to other hepatotoxins such as certain

drugs, alcohol, and chlorinated solvents (ATSDR, 1996b; 1993c, 1990d; Leikin & Paloucek, 1998).

Sulfur Dioxide (Sulfur Oxide)

a. Description

Sulfur dioxide is an unpleasant smelling and highly irritating colorless gas that is soluble in water. It is efficiently absorbed in the upper respiratory tract.

b. Uses

Sulfur dioxide is used in industry to manufacture acid, sulfites and thiosulfates, in mechanical refrigeration, and in agriculture as a bactericide and disinfectant. It is used in the process of food preservation and in the manufacture of kerosene space heaters.

c. Signs and Symptoms

Signs and symptoms include cyanosis, lacrimation, rhinorrhea, choking sensation, caustic dermal burns, corneal injury, chest pains, and cardiac arrhythmias. Inhalation may produce laryngospasm, dyspnea, respiratory distress and death. Concentrations of inhaled sulfur dioxide produce differing effects. Low concentrations produce systemic acidosis, while moderate concentrations produce pulmonary edema. High concentrations produce respiratory arrest. Inhaled sulfur dioxide is slowly removed from the respiratory tract. Individuals with asthma are especially sensitive to sulfur dioxide. Sulfur dioxide poisoning/exposure accelerates atherosclerosis.

There is limited data on the effects of chronic exposure.

d. Emergency Treatment

Treatment is primarily symptomatic. Emergency treatment for sulfur dioxide includes removal of victim from the contaminated area. Do not induce vomiting. Exposed eyes should be irrigated with copious amounts of running water for at least 15 minutes. Exposed dermal areas should be flushed with copious amounts of running water. A patent airway should be maintained while awaiting transport to the emergency room (Leikin & Paloucek, 1998).

Sulfuric Acid (Vitriol, Brown Oil)

a. Description

Sulfuric acid is a colorless, odorless liquid that is extremely corrosive. It is a strong acid and corrosive by nature. Because of its affinity for water, concentrated sulfuric acid dehydrates flesh to the point of charring. Dilute acid injures skin and mucous membranes because of its acidic properties. Routes of exposure include dermal and mucous membrane exposure and ingestion. If ingested, it causes marked injury to the mucosa from the mouth to the stomach causing excruciating pain and swelling of the affected tissue. In addition, salivation, dysphagia, hoarseness, and marked dyspnea may occur. The affected individual can quickly go into shock and cardiovascular collapse.

b. Uses

Sulfuric acid is found in auto batteries, and is used in fur and leather industries, and in the manufacture of acetic acid and hydrochloric acid. Sulfuric acid is also used for metal cleaning and hydrolysis of cellulose.

c. Signs and Symptoms

Signs and symptoms of exposure due to the corrosive effect on skin and mucous membranes include excruciating pain and swelling in the affected area, salivation, dysphagia, hoarseness, marked dyspnea, discoloration of the teeth, corneal and dermal burns, blindness, hemoptysis, hematemesis, shock, and cardiovascular collapse.

d. Emergency Treatment

Remove the patient from the contaminated environment. Emergency treatment consists of dilution of the acid using large volumes of water. The acid is neutralized with baking soda (sodium bicarbonate), cold milk, cornstarch or 15 ounces of cold water. Activated charcoal is not effective. Do not induce emesis because of the corrosive nature of sulfuric acid. Gastric lavage should be attempted if ingestion occurred within one hour previously (Leikin & Paloucek, 1998).

Toluene (Methyl Benzene)

a. Description

Toluene is a colorless liquid with a hydrocarbon derived from coal tar. It is immiscible in water and dissolves fats. The principal source of toluene exposure is the inhalation of gasoline fumes or of indoor air polluted by cigarette smoke and toluene fumes. Intentional inhalation of glues, paints, and solvents results in toluene exposure and produces a feeling of euphoria. Ingestion and dermal contact are also exposure routes.

b. Uses

Toluene is used primarily as a solvent. It is found in glues, paint removers, pesticides, household aerosols, degreasers, adhesives, gasoline and nail polish. Toluene is used in printing, leather tanning and some chemical processes.

c. Signs and Symptoms

Toluene is irritating to the eyes and to the mucous membrane of the nose and respiratory tract. Toluene toxicity includes burning sensation in the mouth and stomach, nausea and vomiting, cough, chest pains, headache, giddiness, vertigo, ataxia, restlessness, euphoria, confusion, stupor, and coma.

The main adverse effect of toluene is CNS depression, producing narcosis and respiratory depression. Cardiac dysrhythmias may occur. Toluene produces a mild macrocytic anemia but no leukopenia. Severe and possibly fatal bone marrow damage may occur as well as late onset of severe blood dyscrasias. Death may occur from respiratory failure or ventricular fibrillation.

Chronic toluene exposures have been associated with headache, lassitude, and nausea. Workers repeatedly exposed have reported loss of coordination, memory loss, and loss of appetite. Some workers
have developed reversible toxic optic neuropathy after chronic exposure in the workplace. Mothers who were exposed to high levels of toluene during pregnancy may have babies with neurological deficits or retarded growth.

Chronic exposure due to solvent abuse can result in permanent neuropsychiatric manifestations. Myopathy, cardiovascular
effects, renal tubular damage, and sudden death have occurred in chronic glue sniffers. Metabolic acidosis can result from renal tubular disorders.

d. Emergency Treatment

Emergency treatment consists of removal of the victim from the contaminated area. HAZMAT personnel are required. Victims should be transported to the emergency room. There is no antidote for toluene poisoning. Emesis is contraindicated because of risk of CNS depression with pulmonary aspiration. A cathartic and activated charcoal may be used. Oxygen and parenteral fluids should also be administered. Monitoring for hypokalemia and acidemia should be done. Epinephrine should be avoided because of the risk of cardiac irregularities. In chronic exposures, evaluate renal and liver function (ATSDR, 1993d; Leikin & Paloucek, 1998).

Vinyl Chloride (Monochloroethylene, VC, and VCM)

a. Description

Vinyl chloride is a manmade chemical that is a colorless gas at room temperature but is normally stored under pressure and used as a liquid. It has a mild sweet odor which, when detected by human smell, is already at a dangerous level. Vinyl chloride is soluble in fats and organic solvents and is slightly soluble in water. Although vinyl chloride released into the air is volatilized quickly, vinyl chloride in groundwater will remain active for months or years. Vinyl chloride is not corrosive to metal when dry but in the presence of moisture, it corrodes iron and steel.

Absorption of vinyl chloride occurs quickly through inhalation, dermal exposure and ingestion. Vinyl chloride is metabolized in the liver. Metabolites can cause hepatic cellular damage or be further metabolized to compounds that are excreted in the urine. Hepatotoxicity risk is increased in persons with prior or concurrent use of alcohol or other chemicals or drugs known to be harmful to the liver. During childbearing years, women should avoid exposure to vinyl chloride which may result in congenital malformation or increased risk of developing cancer in offspring.

Exposure to vinyl chloride may occur through air polluted with vinyl chloride released from hazardous waste sites and through groundwater contamination. Small amounts of contamination may seep into food and liquids packaged in various forms of vinyl chloride. Leaching of small amounts of vinyl chloride monomer into the drinking water supply may occur because of new polyvinyl chloride (PVC) piping.

Tobacco smoke contains a small amount of vinyl chloride.

b. Uses

Vinyl chloride is used primarily in the manufacture of PVC , a plastic used to make pipe, electrical wire and cable coating, flooring, home furnishings, toys, packaging, apparel, automobile parts, upholstery, and aerosol propellant.

c. Signs and Symptoms

Acute vinyl chloride exposure produces CNS symptoms: headache, dizziness, euphoria, ataxia, and narcosis. Cardiac (ventricular fibrillation), circulatory, and respiratory irregularities have been noted. Narcosis may result in death. Short-term exposure to high concentrations of vinyl chloride may be tolerated without lasting effects.

Vinyl chloride is a human carcinogen. Chronic exposure has been associated with hepatocellular injury, malignant and nonmalignant liver tumors, decrease in pulmonary flow, interstitial pneumonitis, and meat wrapper's asthma. Subtle neurologic effects have been observed. Dermal

signs of exposure include a thickening of the skin (characterized by a scleroderma-like condition), pallor, and cyanosis of the fingers. Hepatomegaly, splenomegaly, epigastric pain, thrombocytopenia, and esophageal varices may result.

d. Emergency Treatment

The individual should be removed from the source and brought to the emergency room for supplemental oxygen or gastric lavage as needed. Vomiting should not be induced.

Treatment of chronic exposure is supportive in nature. Exposed individuals should be counseled to avoid alcoholic beverages, tobacco use, further exposure to vinyl chloride, acetaminophen, isoniazid or other hepatotoxins (ATSDR, 1990e; Leikin & Paloucek, 1998).

Xylene (Dimethyl Benzene, Violet 3, Xylol)

a. Description

Xylene is a colorless, sweet-smelling liquid that is highly flammable. It is found naturally in petroleum and coal tar and is formed during forest fires. Xylene is one of the top 30 chemicals produced in this country. Exposure to xylene occurs by inhalation, eye contact, and ingestion. Since xylene is heavier than air, it may cause asphyxiation if exposure occurs in a closed, poorly ventilated, or low-lying area. Sources of exposure include the breathing of xylene in the workplace or near a waste site, in automobile exhaust, and in cigarette smoke. Xylene vapor is irritating to mucous membranes, and eye splashes may cause corneal injury. Prolonged skin exposure will cause defatting of the exposed area resulting in cracking and peeling. Ingestion of xylene through drinking contaminated water can result in acute toxic effects.

b. Uses

Xylene is used as a solvent and in the printing, rubber, and leather industries. It is a cleaning agent, paint thinner, an ingredient in paints, varnishes, gasoline, polymers, glues and in histology laboratories.

c. Signs and Symptoms

Target organs are blood, kidneys, spleen, bone marrow and CNS. Acute exposure causes CNS depression in the form of dizziness, headache, confusion, nausea, impaired gait, and blurred vision. Other symptoms are impaired memory, burning throat, nystagmus, skin irritation, cough, hematuria and syncope. High levels of exposure cause tremors, rapid respiration, paralysis, loss of consciousness, coma, and death. Acid-base imbalances and cardiac irregularities may occur. Respiratory effects include accumulation of fluid in the lungs and respiratory arrest.

Repeated exposure to xylene due to solvent abuse can result in progressive and permanent neuropsychiatric manifestations.
Xylene also can affect the heart, kidneys, and liver and may cause anemia. Xylene is not reported to be carcinogenic. Exposure during pregnancy may result in fetal damage.

d. Emergency Treatment

Emergency treatment requires a HAZMAT team. Secondary contamination of rescue workers can occur. There is no antidote for xylene. Treatment consists of supportive measures. Skin or eye contamination requires washing the affected skin area with mild soap and water or flushing the eye

with copious amounts of water. In cases of ingestion, emesis should not be induced. Stomach contents should be diluted with mild liquid or water. Activated charcoal may be given (ATSDR, 1996c; Leikin & Paloucek, 1998).

Zinc (Zinc Chloride, Zinc Oxide)

a. Description

Zinc is an element found naturally in the earth's crust. Pure zinc is a bluish-white, shiny metal. It is found in air, soil, water, and foods. Zinc combines easily with other elements to form zinc compounds including zinc chloride, zinc oxide, zinc sulfate, and zinc sulfide. Zinc is an essential nutrient required for normal nucleic acid, protein, and membrane cell growth and division. Zinc plays a role in the maintenance of the structure of genes. While zinc deficiency has been associated with health problems, zinc excess has been associated with toxicity. That is to say, ingestion of large quantities of zinc interferes with copper absorption.

b. Uses

Zinc is used in electroplating, smelting, and ore processing and is found in drainage from both active and inactive mining operations. It is used as a component in brass, bronze, die casting, metal alloys, and paints.

c. Signs and Symptoms

Exposure to high levels of zinc occurs when individuals eat food, drink water, or breathe workplace air that is contaminated. Eating large amounts of zinc (10-25 times greater than the recommended daily allowance) can result in stomach cramps, nausea, vomiting, hyperthermia, corneal damage, hypertension, hematuria, shock, and death. Chronic exposure can cause sideroblastic anemia (secondary to zinc-induced copper deficiency), pancreatitis, and lowering of high-density lipoprotein. Breathing large amounts of zinc dust or fumes can cause short-term metal fume fever.

Long-term effects of breathing high levels of zinc are not known.

d. Emergency Treatment

Emergency treatment for zinc exposure includes decontamination and removing the victim from the source of the fumes. Patent airway and breathing should be maintained until the person is transported to the emergency room. Ingestion of zinc should be treated by trained personnel. Vomiting should not be induced if there has been ingestion of zinc chloride or phosphide, due to their corrosive nature. The stomach contents should be diluted with water or milk. Calcium disodium (EDTA) may be used to promote urinary elimination. N-acetylcysteine has been used to promote urinary excretion of zinc sulfate (ATSDR, 1995c; Leikin & Paloucek, 1998).

Learning Activities

Identify contact numbers for your state Poison Control Center.

Assign small groups of students to read selected references that pertain to individual chemicals noting the classification of the hazardous substances, routes of exposure, half-life, mechanism for metabolism and elimination, and toxicities.

Develop separate charts for those chemicals that produce neurotoxic, nephrotoxic, hepatotoxic, immunotoxic, and/or carcinogenic effects.

Summarize the human factors associated with increased sensitivity to hazardous substances such as lifestyle, prior medical condition, diet, and cultural practices.

Have students examine the defining characteristics of the nursing diagnosis High Risk for Poisoning in the NANDA (North American Nursing Diagnosis Association, 1997) Manual. Determine if there are any additions or changes they would recommend to the defining characteristics based on their learning from this chapter.

++Review ATSDR's Case Studies in Environmental Medicine: Nitrate\Nitrite Toxicity (see order sheet in Appendix AA for information on obtaining these materials). Complete the Worksheet in Appendix A of this Module. Identify who in the community is most at risk for health effects from exposure to this substance. Provide a description of the chemical, its uses, where you will most likely encounter the substance (i.e. home, work or school environments), signs and symptoms of acute and chronic exposures, and emergency treatment. What areas or regions in your state are at risk for exposure to this substance? Identify 3 nursing roles for prevention of exposure.

Teaching Methods

Lecture, small group discussion, assigned readings, case study analysis, and patient/community teaching plans.

Evaluation

Students may be evaluated for class participation, small group involvement, poster development, and teaching plan development. Synthesis of information about pesticides, heavy metals, solvents, or carcinogens may be evaluated through short research papers.

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Appendix A: Worksheet for Nitrate/Nitrite

Description of Substance	Uses and Source Locations	Signs and Symptoms of Exposure	Emergency Treatment	Nursing Roles in Prevention